THE PATHOLOGIC ANATOMY OF RUPTURED CEREBRAL ANEURYSMS*

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INCREASING interest in the surgical treatment of cerebral aneurysms suggests that an evaluation of the potentialities of this form of therapy might be aided by a review of some of the anatomic findings in 143 autopsied cases of subarachnoid hemorrhage caused by leaking miliary or berry aneurysms of the circle of Willis.

The sex and age distribution in this group differed significantly from that in cases of apoplexy previously reported. Women predominated 91 to 52; 31 per cent of the patients were under 40 years of age as compared to 9 per cent in the apoplectic group. The youngest patient was 14 and the oldest 94.

The aneurysms were all located above the carotid siphon on the circle of Willis, its peripheral branches or its basivertebral component. Forty-eight involved the anterior communicating artery, 51 the internal carotid, 30 the branches of the anterior, middle or posterior cerebral arteries, and 14 the basivertebral stems. The left half of the circle was involved in 54 cases, the right in 51 and the midline in 38 (Fig. 1).

The aneurysmal sac originated at arterial branchings, although not necessarily at a bifurcation, in 101 cases and by outpouchings from the vascular stem in 42. When the aneurysms involved the origin of an artery, particularly the anterior choroidal from the internal carotid, the distal portion of the vessel was frequently continuous with the fundus of the sac.

Multiple Aneurysms. Multiple aneurysms were present in 27 patients, bilaterally placed in 13 and in the midline as well as on one or both sides in 7. Ruptured and unruptured sacs were found on the same artery in 2, on the

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same side of the carotid stem in 7 and on the basivertebral branches in 7.

Meningeal Hemorrhage. Bleeding into the subarachnoid space was found at necropsy in 137 patients. Evidence of ancient leakage was noted in 6 patients dying of causes unrelated to aneurysmal rupture. Bleeding into the basal cisterns occurred in the majority of cases but was confined to the convexity on the side of the aneurysm in 12, to the median fissure in 7 and localized to the immediate region of the sac in 10. Subdural extension occurred in 22 cases.

Continued leakage or recurrent hemorrhage from the aneurysmal sac apparently occurred in 121 patients. Resolution of the bleeding, manifested by staining of the meninges and hemolyzed blood around the basal structures, was found in only 6 patients, 2 of whom died within 5 days after the onset. Recent bleeding or fresh hemorrhage superimposed on older clots was present in 26 patients who had been hospitalized longer than 1 week and 12 with symptoms of 2 to 6 weeks' duration. Signs of older leakage, obviously antedating the episode of acute bleeding, could be demonstrated in many instances. Bronzing of the meninges, infiltration of tissue around the sac by blood pigment and collections of similar pigment along the basal vessels were noted in 13; envelopment of the aneurysm, wholly or partially, by a pseudo sac in 22; and dense adhesions between the sac and the arteries or adjacent structures in 25.

Parenchymatous Lesions. Destruction of brain tissue, more extensive than erosion or hemorrhage around the sac, complicated the rupture of the aneurysm in 94 cases. Frank hemorrhage occurred in 29 cases, as a subcortical hematoma contiguous to the aneurysm in 21, and deep within the brain structures at a distance from the sac in 8. Focal hemorrhagic or anaemic infarction involving both cortical and deep structures, frequently in more than one area, was present in 65.

Disregarding the nature of the parenchymatous destruction (hemorrhage or infarction), the site of the lesion, in the majority of cases, could be correlated with the location of the aneurysm on the cerebral vascular tree.

Aneurysms arising from both the anterior cerebral and the anterior communicating arteries were associated with damage to the medial portion of the frontal lobe and the corpus callosum, with additional involvement of the orbital portion of the frontal lobe and the caudate nucleus in cases of aneurysms of the anterior communicating artery. The cortex and subcortex of the convexity were involved with aneurysms of the middle cerebral in the Sylvian fissure. Lesions in this area, as well as of the lenticular region and the subependymal tissue around the inferior horn of the ventricle were present with aneurysms of the internal carotid. Destruction within the cerebellar hemisphere and damage to the retro-olivary portion of the medulla, respectively, was noted with two aneurysms arising from the basivertebral stem, one at the origin of the superior cerebellar and the other at the origin of the posterior inferior cerebellar. Lesions of the midbrain and upper pons occurred with aneurysms of the divisional branches of the basilar.

In general, the parenchymatous damage associated with leaking aneu-
Aneurysms occurred in the territory of stem vessels located distal to the sac. It would seem, therefore, that circulatory disturbances in the vascular bed of the peripheral field of supply resulting from aneurysmal rupture must be considered as a factor in the production of the brain lesions. However, lesions confined totally or in part to the distribution of ganglionic arteries were associated with 10 aneurysms of the middle cerebral in the Sylvian fissure, and bilateral lesions of the globus pallidus with unilateral destruction of the cortical grey ribbon of the island of Reil with an aneurysm of the anterior communicating. In view of these findings, other factors, such as rapidly increasing intracranial pressure, may also contribute sufficient circulatory disturbance to produce tissue damage in susceptible areas.

**Extraneural Disease Associated with Leaking Cerebral Aneurysm.** We have previously reported that, although aneurysms were present in 131 of a series of 1,335 brains examined at necropsy, leakage or rupture was present in only 37 (28 per cent). Little attention has been paid to the factors that precipitate bleeding from cerebral aneurysms. The etiologic role of cardiovascular or other chronic organic somatic disorders is difficult to evaluate in elderly patients. It seems worthy of mention, however, that hypertrophy of the heart, particularly of the left ventricle, was present in 87 of the group of 40 patients under 40 years of age. Dissecting aneurysm of the aorta, severe coronary sclerosis or fibrosis of the myocardium were present in 3 patients, the oldest of whom was 50. Rheumatic valvular disease was noted in 3, pericardial effusion in 2 and adhesive pericarditis in 1—all under 40 years of age. In this younger group, subarachnoid bleeding occurred during acute pulmonary tuberculosis in one patient and during a crisis of sickleemia in another.

Of the 97 patients over 40 years of age, 41 were under treatment for cardiac disorders prior to the onset of the subarachnoid bleeding; 15 additional instances of luetic or rheumatic heart disease were found post mortem. Patent foramen ovale was noted in 3 cases, pericardial effusion in 1 and a large aneurysm of the aortic arch in another. Subarachnoid bleeding occurred in 3 chronic alcoholics during a debauch and in 1 patient at the onset of diabetic acidosis.

**Associated Anomalies.** Extracranial anomalies and malformations have frequently been reported as associated with cerebral aneurysms but were present in only 9 patients in this group. Patent foramen ovale or fenestration of the aortic valves was noted in 4, polycystic disease of the kidney in 1, and horseshoe kidney in another. Hypoplasia of one kidney or one adrenal was noted twice, reduplication of the ureter, once.

Anomalous formation of the circle of Willis in the presence of cerebral aneurysm, however, was present in 118 of the 124 cases in which complete description was available. Because of the relatively few cases in which the posterior half of the circle of Willis was involved, analysis of the intracranial anomalies associated with leaking aneurysms is limited to 119 cases in which the aneurysmal lesion involved the internal carotid or its branches. Essentially, the anomalous formation resulted from hypoplasia of one or more of
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FIG. 2. Variations in circle of Willis associated with 114 cases of ruptured miliary aneurysm.
I. Normally formed circle of Willis—10 cases.
II. Hypoplasia of all stem vessels which are relatively equal in size (foetal type)—6 cases.
III. Hypoplasia of the first portion of one anterior cerebral artery—47 cases.
IV. Hypoplasia of the divisional branches of the basilar artery on both sides, with origin of both posterior cerebral arteries from the internal carotid stem—11 cases.
V. Hypoplasia of the divisional branch of the basilar artery on one side, with origin of the posterior cerebral artery of the same side from the internal carotid stem—20 cases.
VI. Hypoplasia of the first portion of the anterior cerebral artery and of the divisional branch of the basilar on the same side with the posterior cerebral artery of that side originating from the internal carotid stem—15 cases.
VII. Hypoplasia of the first portion of one anterior cerebral artery, and of the divisional branch of the basilar on the opposite side, the posterior cerebral artery originating from the internal carotid stem on the side of the hypoplastic divisional artery—6 cases.
VIII. Hypoplasia of the first portion of one anterior cerebral artery, and of the divisional branches of the basilar on both sides, both posterior cerebral arteries originating from the internal carotid stem—5 cases.
Fig. 3. Silhouettes of the cerebral vascular tree in ruptured aneurysm.

**Top row (L–R)**


2. Aneurysm terminal bifurcation 2nd portion anterior cerebral. Second aneurysm bifurcation opposite middle cerebral artery. Hypoplasia one divisional branch of basilar; posterior cerebral from internal carotid.

3. Aneurysm anterior communicating artery. Recurrent artery of Huebner from sac. Extreme hypoplasia first portion one anterior cerebral artery, hypoplasia divisional branch basilar; posterior cerebral from internal carotid.


**Middle row**


6. Aneurysm internal carotid distal to posterior communicating. Second aneurysm bifurcation of middle cerebral artery same side. Hypoplastic circle of Willis with hypoplasia of divisional branches of basilar; posterior cerebral arteries from carotid stem.

7. Aneurysm internal carotid artery distal to posterior communicating. Anterior choroidal from sac. Hypoplasia divisional branches basilar; posterior cerebral arteries from internal carotid stems.


**Bottom row**

9. Two aneurysms of divisional branch basilar, proximal to junction of posterior communicating. Normally formed circle.


12. Wide-necked aneurysm involving internal carotid and posterior communicating. Hypoplasia anterior communicating and one divisional branch basilar; posterior cerebral artery from internal carotid stem.
the component vessels, reversion to an embryonic stem of origin, or a combination of these factors. Eight basic patterns could be identified (Figs. 2 and 3).

No absolute correlation of aneurysms of a particular location with specific anomalous formation was found although 85 per cent of the 40 aneurysms of the anterior communicating artery were associated with hypoplasia of the first portion of one anterior cerebral, and the great majority of the aneurysms present lay in locations where circulation would be influenced by focal increase of resistance or altered field of supply.

DISCUSSION

The results of surgical treatment for intracerebral aneurysms are based admittedly on carefully selected cases, particularly those in which focal signs, such as a third nerve palsy, precede any evidence of frank bleeding. The patients in our series represent an unselected cross section from a large general hospital and, with the exception of 6 patients, were admitted with signs indicative of intracranial bleeding. All aneurysms were located above the level of the sella. Even in selected cases, the best surgical results are obtained when the aneurysm is located at the carotid siphon. Surgical trapping in our cases would have been obviously dangerous because of the location of the aneurysm in reference to the production of permanent neurologic defects such as hemiplegia, and secondly, because of the difficulty in establishing adequate collateral circulation in an anomalously formed circle of Willis.

In such cases, a number of factors require thoughtful consideration before surgical therapy can be recommended. The general cardiovascular complications found frequently even in the younger age group are a potential source of danger, as is the presence of multiple aneurysms in approximately 20 per cent of cases. Concomitant parenchymatous lesions in 94 also limit the possibilities of successful surgery since the prognosis is poorer in cases accompanied by intracerebral extension than in those in which the hemorrhage is confined to the subarachnoid space.

SUMMARY

The patho-anatomic findings in a series of 143 cases of subarachnoid hemorrhage caused by leaking miliary aneurysms have been evaluated in relation to the potentialities for successful surgical intervention. All aneurysms lay above the carotid siphon; 129 originated from the internal carotid stems, and 14 from the basivertebral. Multiple aneurysms were present in approximately 20 per cent of the cases. Evidence of repeated bleeding, either ancient or recent, was present in most cases. Parenchymatous lesions were present in 94 cases, but hemorrhage accounted for only 29. Somatic disease, chiefly involving the cardiovascular system, complicated many cases. Anomalies of the circle of Willis were extremely common and of such a nature as to mitigate against adequate collateral circulation. Careful consideration of these factors is necessary in evaluating the potentialities of surgical therapy.
REFERENCES